THE QUESTION OF WATER AND ELECTROLYTE LEVELS AT HIGH TEMPERATURES

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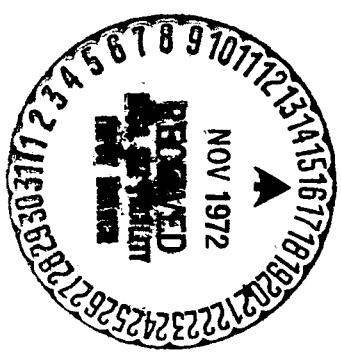
Translation of: "Zur Frage des Wasser und Elektrolythaushaltes bei hohen Temperaturen," *Arbeitsmedizin-Sozialmedizin-Arbeitshygiene*, Voo. 2, 1966, pp. 44-50.



NATIONAL AERONAUTICS AND SPACE ADMINISTRATION
WASHINGTON, D.C. 02546 OCTOBER 1972

NASA TT F-13,915

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ABSTRACT. Physiological regulatory mechanisms governing the water and electrolyte levels in the body are studied, together with the various disturbances caused during work at high temperatures.

Water comes second after oxygen in the list of vitally important substances, /44* and this is suprising because water is the largest individual building block of the body as far as quantity is concerned. In addition, healthy individuals show practically no variations in body water, although its intake and excretion can vary significantly.

As far as the amount of body water is concerned, the figure of 90% that goes back to Claude Bernard has proven unreliable. More recent studies show that the water content of the adult human being varies between 54% and 66%. Three-quarters of the total body water is contained in the skin and muscles, while only 10-30% is found in the water-poor fat tissue. The fat tissue can make up 10-50% of the body weight, so that the percentile water content of an organism is dependent upon its fat content. According to Behnke as well as Rathburn and Pace, every normal body has a basic structure with normal composition ("lean body man"), the so-called "fat-free body mass." If this basic structure has fat deposits superimposed on it, the percentile component of the body water decreases with increasing weight, but its absolute quantity largely remains constant.

*Numbers in margin indicate pagination in the foreign text.

All of the body water is distributed between two spaces: the intra- and extracellular fluid spaces.

The metabolic processes of the cells take place in the intracellular water, but the composition and volume remain constant in the face of external disturbances.

The extracellular water differs in composition and volume significantly from the intracellular water. Complicated regulatory mechanisms manage to keep it as steady as possible. The extracellular fluid is divided into interstitial and intravasal water, with the volume of the latter corresponding to the plasma volume. Figure 2 shows the electrolyte distribution in the fluid spaces.

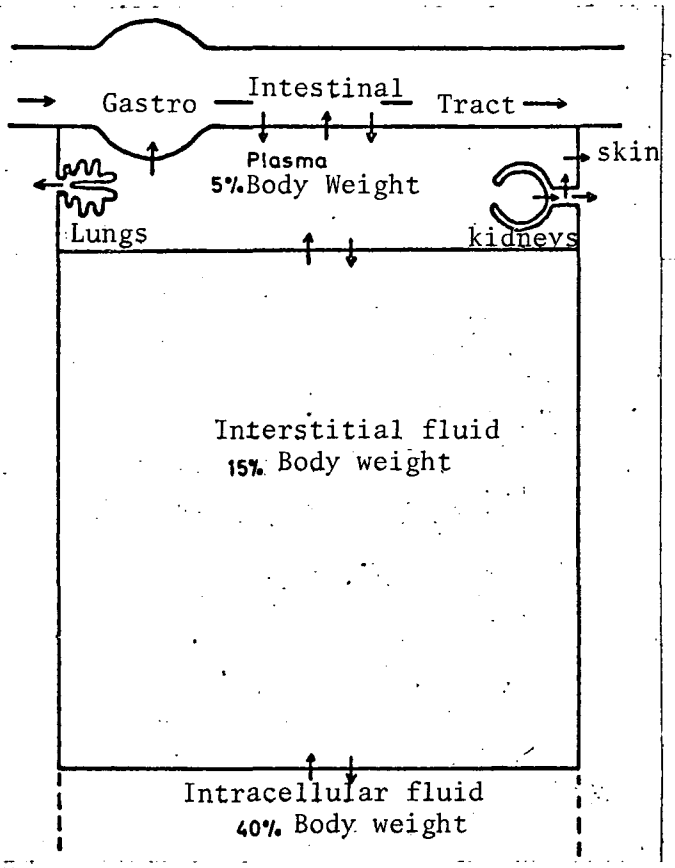

Figure 1. The Fluid Spaces of the Body, Their Exchange Possibilities and Size (After Gamble).

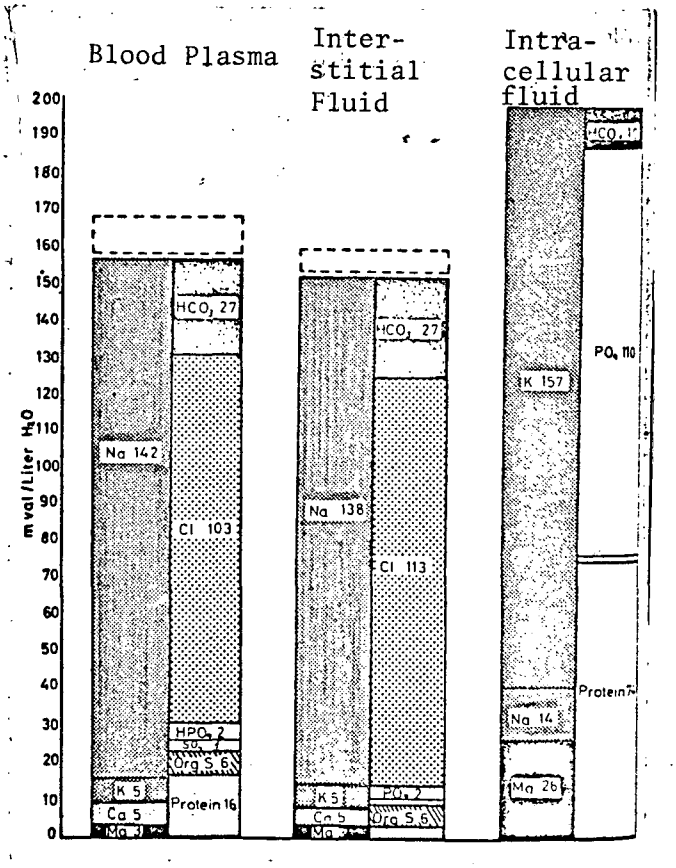

Figure 2. Distribution of Electrolyte in the Fluid Volumes of the Body (After Gamble).

In the extracellular space, Na^+ predominates as the cation, while Cl^- and HCO_3^- are the anions. The plasma and interstitial fluid are practically the same in composition, and the slight differences that do exist due to the higher protein content of the plasma can be explained by the Donnan equilibrium. In the intracellular fluid, on the other hand, K^+ and Mg^{++} are found as the principal cations and the anions are protein and HPO_4^{--}. /45

98% of the total potassium is found in the cells, 60% of the sodium is in the extracellular space. This creates an ionic imbalance, for whose maintenance energy expenditure is required by the cells: sodium (and water with it), which flows into the cells along the concentration gradient must constantly be pumped out again from them. This is performed by the "sodium pump" - the presence of a water pump is debatable.

The ionic concentration gradient between the extra- and intracellular fluids, as a membrane potential, acts as a precondition for the excitability of nerves and muscle cells. If energy formation is disrupted by problems invovling glycolysis, the citric acid cycle of the oxidative phosphorylization the sodium pump collapses. The result is the penetration of sodium and water into the cells with simultaneous escape of potassium into the extracellular space. Hence, the regulation of the water and electrolyte levels is of critical significance for the maintenance of life.

Water can reach the organism in three ways:

1. In the form of potable fluids,

2. With solid foods (meat contains about 75% water and vegetables 90%),

3. The burning of fats, carbohydrates and proteins leads to the production of water of oxidation (1 g of fat = 1.07 ml H_2O; 1 g carbohydrate = 0.56 ml H_2O, and 1g protein = 0.4 ml H_2O).

The loss of water can occur in the following ways:

1. Invisible water loss through the lungs. This occurs without loss of electrolyte and depends on energy conversion. The values are in the vicinity of 15 ml per 100 calories, i.e., approximately 300 ml per day;

2. Through the skin, likewise invisibly and without electrolyte loss. Persons who are not perspiring can lose 600 ml per day in this fashion, with an energy expenditure of approximately 30 ml per 100 calories.

3. In the stool. Only small amounts of water are eliminated in this fashion, approximately 200 ml per day.

These three types of fluid loss are obligatory, i.e., they occur even if the organism is not taking in water. A fourth possibility for fluid loss is provided by the kidneys. In contrast to the three other methods, the amount of urine excreted can vary: with a daily average of 1500 ml, variations from 300 ml to several liters per day are possible. Likewise, the amount water lost through perspiration can vary. The normal water balance of an adult is shown in Figure 3.

The electrolyte requirement of a normal individual is shown in Figure 4.

I. Input		II. Output	
Oral fluid	1300 ml	urine	1500 ml
Fluid in solid food	1000 ml	feces	200 ml
Water of oxidation		skin	600 ml
(from metabolism)	300 ml	lungs	300 ml
	2600 ml		2600 ml

Figure 3. Water Balance in the Adult (After Klaus).

	Minimum	Maximum	Average
Na^+	20	425	100-200 (= 2-5 g Na)
K^+	20	425	50-100 (= 2-4 g K)

Figure 4. Electrolyte Requirements Per Day in mval (After Klaus).

The intake of the electrolytes occurs with the food in the form of inorganic salts, with the intake of the minimum daily requirement usually being exceeded. Even in a starving condition, there is no shortage of electrolyte since the electrolyte is liberated from tissue that is consumed suffice. On an ordinary diet, the adult human being obtains 100 to 200 mval sodium (2-5 g) and 50 to 100 mval potassium (2-4 g). There are three ways in which electrolytes can be lost:

1. With the feces, with vary little normally being lost in this fashion: 2 mval (50 mg) sodium and 5 mval (200 mg) potassium.

2. Elimination of electrolytes with the urine is governed by the intake. Sodium-chloride free food can drop the loss to 1 mval sodium (23 mg) and 1 mval chlorine (35 mg), and in the case of a potassium-free diet the potassium content of the urine drops to 8 mval (300 mg). The average values of the renal electrolyte loss are given in Figure 5.

	mval/l Urine	mval/ Day
Na^+	90	130 (100-200)
K^+	50	75 (60-90)
Ca^{++}	5	7,5
NH_4^+	37	55
Cl^-	100	150 (100-200)
$H_2PO_4^-$	25	37
SO_4^{--}	40	60

Figure 5. Renal Electrolyte Excretion (After Holtmeier).

3. Very large amounts of sodium, chlorine and potassium can be lost with the perspiration. Aldosterone definitely plays a role in this instance, analogous to that of the kidneys: increased aldosterone activity reduces sodium elimination and raises potassium elimination. With comfortable environmental temperature and mild work, approximately 12-25% mval of sodium (250 to 500 mg) are lost with the perspiration and 7 mval (270 mg) potassium per day.

In order to keep the total body water and electrolytes at constant value, /46 the water and electrolyte are under the control of regulator mechanisms that operate in a very precise fashion. It is obvious that there are very close relationships between the water and electrolyte levels.

The intake of water is regulated by the feeling of thirst. This is understood to be consciousness of a need for water intake. A shortage of water leads to a rise in the osmotic pressure of the extra- and intracellular fluid with simultaneous decrease in this fluid volume. The *simultaneous* rise in the osmotic pressure in the extra- and intracellular spaces does not produce thirst, however (see urea delivery). A shortage of water can only affect the decrease in the fluid spaces. For example, if the extracellular space becomes smaller due to a loss of isotonic fluid (hemorrhage, diarrhea), thirst results as well as a rise in the osmolarity of the extracellular fluid, in which case the intracellular fluid is reduced by flowing into the extracellular

space. In case there is no change in the osmolarity of the extracellular fluid, a shortage of extracellular or intracellular fluid will cause thirst.

A drop in the extracellular volume also occurs in the event of a shortage of sodium chloride, without a regular feeling of thirst resulting in this instance. In this case, the osmotic pressure in the cells rises, so that water is displaced into them and thus increases the intracellular fluid volume.

Local dryness of the mouth and throat as well as the flow of saliva play hardly any role in the development of the sensation of thirst.

Figure 6 shows a diagram of the factors which are responsibile for the development of a sensation of thirst.

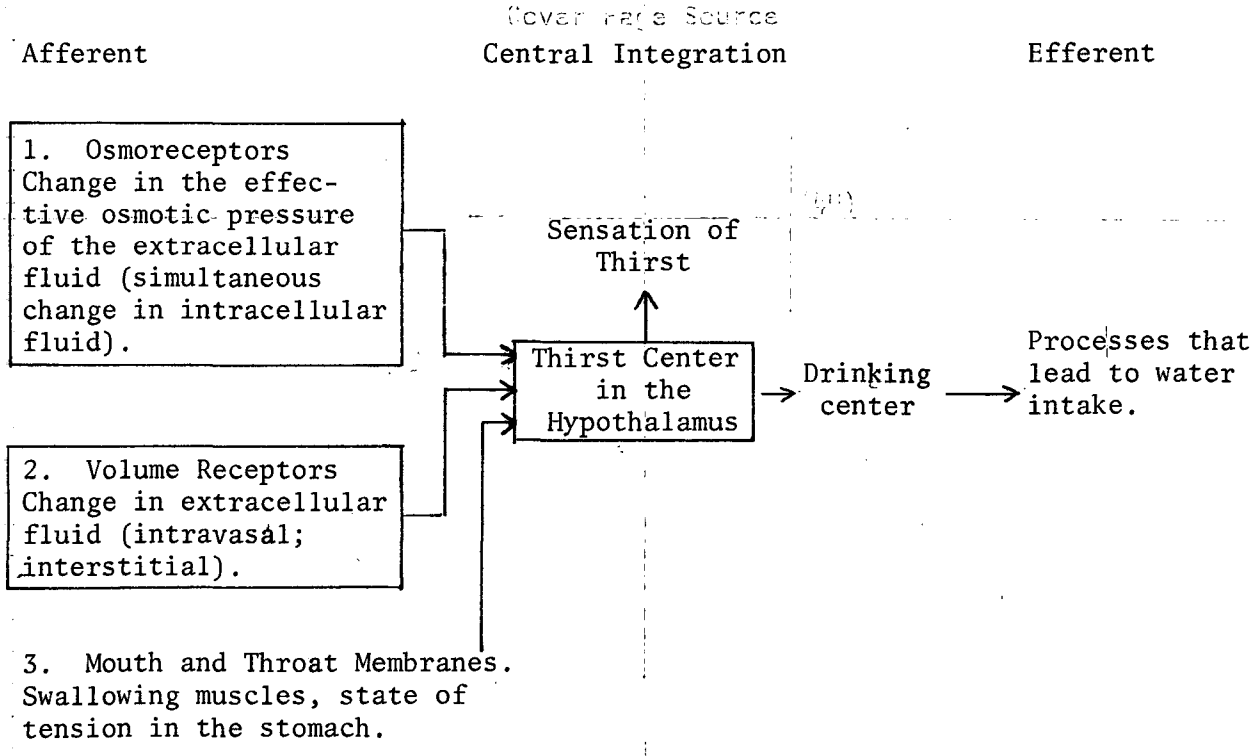

Figure 6. Schematic Representation of the Critical Factors Responsible for the Development of a Sensation of Thirst (After Schwab and Kuehns).

In the vicinity of the supraoptico-hypophyseal system there are areas in which the integration of the afferent factors producing thirst takes place. It is still uncertain whether a drinking center which controls the motor activity of drinking exists in addition to this thirst center.

The organ responsible for the regulation of water as well as electrolyte level is the kidney alone. Its function is established by a number of regulatory mechanisms mainly for the purpose of keeping the internal environment constant.

One of its most important tasks consists in maintaining isotony, i.e., the normal osmotic concentration in the extracellular space. The control of this process is accomplished by means of osmoreceptors in the vicinity of the internal carotid artery (Verney et al). An increase in osmotic pressure by only 1% will lead to excretion of ADH (andiuretic hormone), which was called arginine vasopressin by du Vignaud. ADH leads to increased water resorption in the kidney tubules. On the other hand, hypotony in the extracellular space leads to an inhibition of vasopressin excretion from the posterior lobe of the hypophysis and hence to increased water excretion. It is important that only changes in the *effective* osmotic pressure have effects of this kind. After administration of urea, which quickly and easily penetrates all the cells and simultaneously raises the osmotic pressure inside and outside the cells to an equal extent, the osmoreceptors do not act.

ADH is also of importance with respect to isovolemia, i.e., for the maintenance of the volume in the extracellular space at a constant level. Gauer et al. have found volume receptors in the area of the left auricle. Any increase in the volume and pressure in the left auricle acts through the hypothalamic-hypophyseal system to produce an inhibition of vasopressin secretion and an increased diuresis (e.g., even under the influence of cold). On the other hand, any drop in the pressure and volume in the left auricle leads to inhibition of diuresis due to a rise in ADH liberation. These volume receptors detect intravasal volume changes exclusively, and do not respond to interstitial ones. A general view of the regulator mechanisms of the water level is shown in Figure 7.

While the water level is mainly kept in equilibrium by ADH, the regulation of the sodium level appears to be due to other mechanisms. Sodium and the corresponding anions (chloride and bicarbonate) determine the effective osmotic pressure in the extracellular space through their concentration. Unquestionably /47 it is sodium which plays the principal role. The processes that regulate their sodium level occur primarily in the area of the tubular epithelium. In the glomeruli, 25,000 mval Na^+ are filtered every 24 hours, but only 100-250 mval appear in the urine. This means that more than 99% are retained, 7/8 of them in the proximal tubule and 1/8 in the distal. The hormones of the adrenal cortex are of critical significance for these processes inasmuch as they liberate the following steroids into the bloodstream: cortisone, aldosterone, and corticosterone (see Gross).

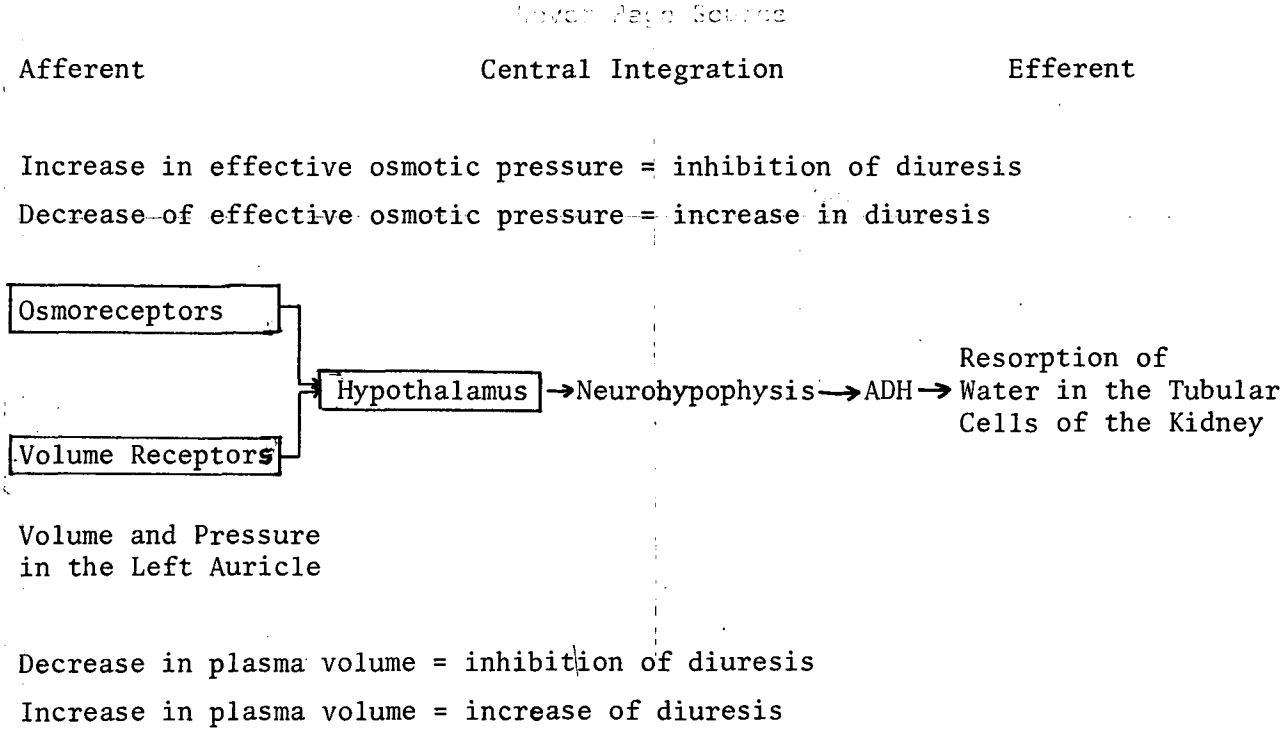

Figure 7. Schematic Representation of the Factors Which are Important for the Regulation of the Water Level (After Schwab and Kuehns).

Lack of hormones from the adrenal cortex leads to a drop in the extracellular sodium concentration (as well as in the chloride and bicarbonate concentration) with a simultaneous rise in the amount of potassium in the extracellular space.

The sodium loss can be attributed to an increased renal sodium elimination due to a lack of resorption in the tubules (Flanagan et al.; Stern et al.). At the same time, more sodium is lost in the perspiration and saliva. These sodium losses can be offset by the administration of aldosterone (as well as corticosterone and cortisone). In the case of severe sodium chloride excess, however, aldosterone alone does not suffice for compensation. Perhaps the hormone from the adrenal cortex which eliminates sodium that was found by Neher et al. in 1958 must also be present. Moreover, in the absence of this hormone electrolyte metabolism can only be completely normalized if cortisone is also given as well as aldosterone, as Rosenbaum et al. as well as Epstein were able to show. Hence, cortisone is also necessary for the normal function of the tubules. Aldosterone and cortisone are probably not the principal cause of acute changes in the sodium level. According to Ingle, they appear to be responsible instead for the basic mechanism of Na resorption, while the exchange of Na for H ions is controlled by aldosterone and that of Na^+ for K^+ is controlled by corticosterone (Morel).

Aside from highly acute states, aldosterone may be the principal factor for sodium resorption in the kidney tubules.

Aldosterone formed in the glomerular zone of the adrenal cortex independently of ACTH (adrenocorticotrophic hormone). Rauschkolb and Farrell were able to show that certain areas in the hypothalamus are important for the formation of aldosterone, and Farrell even named the concept of the "glomerulotropic hormone." The following factors are responsible for the liberation of aldosterone:

1. Limitation of sodium intake. The Na restriction which develops also leads to a reduction of the amount of extracellular fluid, which is also a stimulus to aldosterone secretion.

2. Adminstration of potassium leads to a rise in aldosterone excretion while a shortage of potassium leads to a drop in the latter parameter. In this context, the changes in the potassium level (more potassium increases sodium elimination, less reduces it) appears to affect the sodium level directly, so that the aldosterone secretion in this case is controlled in a roundabout fashion via sodium. The absolute potassium level in the plasma evidently is of no significance.

A shortage of water in the intracellular space, according to Bartter, leads to only a slight liberation of aldosterone. The same takes place during simultaneous work and perspiration. If a shortage of salt exists beforehand, a rise will take place.

In contrast to this, changes in the extracellular fluid are always associated with changes in aldosterone secretion: a rise leads to a decrease in aldosterone liberation and a decrease leads to a rise in the latter. According to Bartter et al., the intravasal volume is solely responsible for this process.

The acute changes in sodium excretion cannot be understood in the light of this theory. Smith has hypothesized a previously unknown humoral factor X to explain such acute changes, which is supposedly formed in the hypothalamic region (by analogy to the regulation of the water level). In addition, the physiological significance of the "natriuretic" hormone (Neher), which is definitely of importance in connection with the development of generalized edema.

It has not yet been determined conclusively whether, by analogy to the volume receptors in the left auricle which influenced the water level, there are also volume receptors which regulate the electrolyte level. Some findings appear to indicate this. Strauss was the first to express the opinion that such volume receptors which influenced the sodium level must be located in the anterior segments of the interstitial space. (See: anti-natriuresis in orthostasis and with reduction of the interstitial fluid following hemorrhage; differential effect of sodium chloride infusions in sitting and lying subjects; /48 dilation of the extracellular space by means of iso- hypo- and hypertonic solutions causes increased sodium excretion.) Without their location being definitely determined, the presence of volume receptors which influence the sodium level is considered today to be relatively certain. They are probably located in the cranial interstitial space, and possibly even intercranially. Using the representation of Schwab and Kuehns, Figure 8 shows how the regulation of the sodium level could be represented.

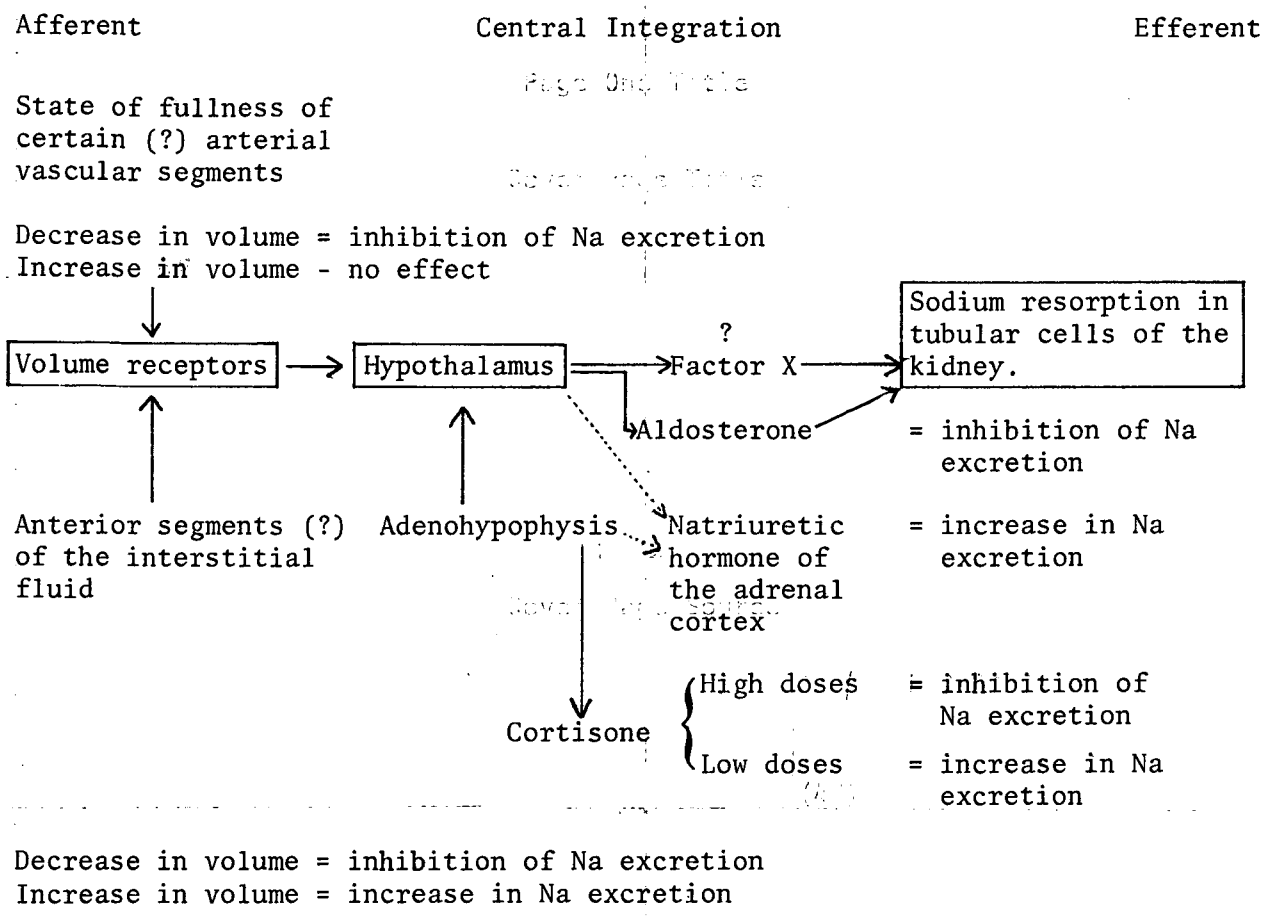

Figure 8. Schematic Representation of the Factors Significant for the Regulation of Sodium Level (After Schwab and Kuehns).

The disturbances of the water and electrolyte levels may be divided into two groups according to their pathogenesis: distribution and balance disturbances.

Distribution disturbances are characterized by changes in the ionic lack of equilibrium between the intra- and extracellular fluid. Administration and excretion are primarily normal in this situation. The cause is a displacement of the acid-base equilibrium as a consequence of disturbances of metabolism in general illness.

Description	Cause	Effect							
		Intra-Cellular Space	Extra-Cellular Space	Intra-Vasal Space	Na^+ in the Plasma	Hema-tocrit	Avg. Ery.* Vol.	Ery.*	Hb.
Hypertonic Dehydration	Water Deficit Removal of more water than sodium	-	-	-	+	(+)	-	+	+
Hypotonic Dehydration	Sodium Deficit Removal of more sodium than water	+	-	-	-	++	+	+	+
Isotonic Dehydration	Removal of water and sodium in an isotonic ratio	N	-	-	N	+	N	+	+
Hypertonic Hyperhydration	Addition or re-tention of more sodium than water	-	+	+	+	--	-	-	-
Hypotonic Hyperhydration	Water Poisoning Addition or re-tention of more water than sodium	+	+	+	-	(-)	++	-	-
Isotonic Hyperhydration	Edema Removal of water and sodium in an isotonic ratio	N	+	+	N	-	N	-	-

*Erythrocytes

Figure 9. Categorization of Disturbances of Water and Sodium Metabolism.

However, the balance disturbances are of greater significance for the subject at hand, which naturally can turn to distribution disturbances especially if they last a long period of time. The cause of balance disturbances is a shortage or excess of water and electrolytes, which can be attributed to /49 changes in their administration or excretion. Balance disturbances of this kind always lead to changes that affect the extracellular space first.

In all six possible cases, three of these disturbances involve an increase in total water content while the other three involve a decrease in it. In the extracellular space, in each of these balance disturbances, the sodium concentration may be normal, increased or decreased. Since sodium is critical for the osmotic concentration, we can speak of the isotonic, hypertonic and hypotonic dehydration or hyperhydration. Figure 9 gives a categorization of the possible disturbances. In this table, there are several characteristic parameters which are of diagnostic value: the behavior of the intracellular, extracellular and vasal space, Na^+ in plasma, hematocrit, average erythrocyte volume, erythrocytes and hemoglobin.

Both concepts--dehydration and hyperhydration--refer only to the extracellular fluid volume. The intracellular fluid volume may behave differently.

Of the different state of dehydration and hyperhydration, three are of significance as far as disturbances of water and electrolyte level are concerned which may occur during work in high temperatures.

1. Lack of water (hypertonic dehydration),
2. Lack of sodium (hypotonic dehydration), and
3. Water poisoning (hypotonic hyperhydration).

It is extraordinarily difficult to calculate exactly the water and electrolyte losses. However, the deficit may be estimated roughly with the aid of the formulas developed by Elkinton and Danowski, Schroeder and Perry and Moll, Stickler and Daugherty (see Figure 10).

Extracellular H_2O Deficit =

$$\frac{Na_{real} - Na_{theoretical}\ (142)}{Na_{theoretical}\ (142)} \times \frac{\text{Theoretical Body Weight}}{5} \text{ (kg)}$$

Total water deficit = extracellular water deficit × 3

Sodium deficit (mval) =

$$(Na_{theoretical}\ (142) - Na_{real}) \times \frac{\text{Body Weight}}{5} \text{ (kg)}$$

Figure 10.

Before the disturbances involved here are discussed, we must consider the possibilities of water and electrolyte excretion through the skin and sweat glands. In addition to the glandular water loss through the sweat glands, there is also extraglandular loss. This involves diffusion of water vapor from the skin capillaries through the outermost layers of the skin, which takes place according to the laws of diffusion (vapor pressure differential) (Buettner).

Considerable amounts of water and salt can be lost through the sweat glands. It should be emphasized, however, that the secretion of perspiration does not constitute a regulatory process for water and electrolyte levels. Rather, it serves exclusively for purposes of heat regulation. Its significance in the latter context will be clear if we analyze the situation in which individual born without sweat glands find themselves. Such individuals, by means of extraglandular loss of 20-30 g of water per hour are capable of elimination only 25% of the resting heat formation (List) and in summer or when performing light work are unable to maintain their body temperature (Sundermann). The water loss through the skin may be very small and is limited at the lower end by the laws of water diffusion. On the other hand, it can be very high, with values up to 4 liters per hour. The upper limit is set by the efficiency of the sweat glands.

Normally the perspiration is hypotonic with respect to the plasma (0.2 - 0.3%). It contains primarily sodium chloride. The salt concentration of the perspiration can also vary as a function of various factors such as heat stress, situation, acclimatization, acquiring values between 0.03 and 0.85%, i.e., the perspiration can be practically isotonic with respect to the plasma.

The amount of perspiration which an individual can produce in a short period of time can amount to 4 liters per hour. The total daily output of 18 liters per 24 hours has hardly ever been exceeded (Eichna et al.; Ladell). The loss of such large volumes of perspiration for a long period of time is impossible, since the sweat glands become fatigued (Robinson). However, 10-12 liters of perspiration per day (according to Lehmann) are not so unusual.

Large amounts of sodium chloride can also be lost with the perspiration. In extreme cases, the loss can amount to 3 g per hour or 20 g in 24 hours (Robinson), i.e., dry heat can lead to a severe impoverishment of the blood with respect to NaCl (Robinson).

If these facts are compared to the physiological and pathological-physiological bases of the regulatory processes for water and sodium, the following pathological states can be seen as likely to occur during work at high temperatures:

1. Water deficit, i.e., hypertonic dehydration. This sort of problem arises in the event of reduced water intake and/or increased water losses. In these cases, we can also speak of thirst exsiccosis. Mariott differentiates between mild water impoverishment (2% of body weight) in moderate (6% of body weight) as well as acute (10% loss of body weight). Loss of 40% of the body weight (25% weight loss) is fatal.

From the pathophysiological standpoint, a shortage alone can lead to a rise in sodium concentration in the extracellular space, i.e., the osmotic pressure increases. Consequently, water is drawn out from the cells, thus leading to a further increase in the osmotic pressure in the cells themselves. This means that the intracellular volume is more sharply reduced than the extracellular volume.

The increase of the osmotic pressure in the extracellular fluid stimulates the osmoreceptors, ADH is excreted and tubular water resorption rises. In very severe cases of water loss, which produce a drop in plasma volume, the heart-time volume decreases. The consequences are reduced blood flow through the kidneys and a drop in glomerular filtration. Due to the escape of water from the cells, the sodium concentration in the plasma is usually only slightly increased and the hematocrit is only slightly above normal due to the shrinkage of the erythrocytes. However, the erythrocyte concentration increases. If hypertonic dehydration occurs as a consequence of profuse perspiration resulting in a water loss which is much greater than the simultaneous salt intake, the /50
rise in the effective osmotic pressure will lead to a pronounced feeling of thirst and those afflicted will drink large amounts of frequently salt-free fluid. If the water intake is sufficient to cover only the fluid loss, the sodium concentration will remain normal and an isotonic dehydration will be found in which circulatory symptoms predominate. On the other hand, if too much water is drunk, the results will be hypotonic dehydration, a state which is characterized by predominant (relative) sodium deficit which in individual cases can even lead to water poisoning.

2. The causes of hypotonic dehydration, the shortage of sodium, can be found mostly in renal or extrarenal sodium losses. In serious cases, the sodium concentration can drop to 130 or even 115 mval/l. Here, the water loss affects only the extracellular volume, so that early symptoms appear in the circulation: dizziness, collapse when standing up. On the other hand, there may be cramps in the muscles being used. There is hardly any feeling of thirst in this situation. Even in extrarenal sodium losses, i.e., during perspiration, the urine volume initially remains normal but hardly any NaCl can be found in the urine.

From the pathophysiological standpoint, sodium deficit leads to a drop in the osmotic pressure in the extracellular space. The drop in osmolarity is initially answered by a rise in diuresis. However, if the extracellular fluid volume has dropped to a certain extent, stimulation of the volume receptors liberates ADH and water is then retained by the kidneys. The result is a

further drop in the osmotic pressure in the extracellular space, leading to an influx of extracellular fluid into the intracellular space: the cells swell. Then, however, there is no further drop in the extracellular volume.

3. Hypotonic hyperhydration is a consequence of excessive water intake. It is observed when electrolyte-free fluids are taken in excess following serious sodium chloride losses, such as those caused by profuse sweating. This can lead to a very serious situation from a clinical standpoint, characterized primarily by somnolence, nausea and emesis. Acute muscular cramps develop, as well as increased flow of tears and saliva as well as diarrhea. The initially large urine volume rapidly drops and anuria may develop. Only rarely are there cases of pronounced edema, primarily in the eyelids. If such a state develops very rapidly, it is called water poisoning. The sodium concentration in the plasma decreases. In contrast to hypotonic dehydration, it is not sodium deficit which is involved here but a dilution-hyponatrèmia. From the pathological-physiological standpoint, an excess of water leads to hypotonia as well as a rise in volume in the extra- and intracellular space.

Only a few brief comments are in order with regard to the therapy of these pathological states:

1. The treatment of water deficit can be performed by administering water orally in mild and moderately serious cases. In serious cases the intravenous administration of isotonic glucose solution is advised in amounts of 3-5-10 liters. After the fluid has been replaced, the replacement of the potassium and the phosphate can be considered, which in the case of long periods of thirst may also be lost.

2. The sodium deficit situation, i.e., hypotonic dehydration is best treated by administration of hypertonic solutions of sodium chloride (50-100-150 mval).

3. In order to relieve a hypotonic hyperhydration or water poisoning, 50-100 ml of a hypertonic sodium chloride solution (5.85%) is administered intraveneously. The symptoms that lead cellular swelling then rapidly diminish.

A warning should be given against the administration of too much sodium chloride, since it can lead to dilation of extracellular space with pulmonary edema. After a level of 130 mval sodium per liter has been reached, the administration of NaCl can be terminated.

An effort has been made in the present paper to present the physiological regulatory mechanisms of the water and electrolyte levels. In addition, a survey is given of the disturbances that can arise during work at high temperatures due to water and electrolyte losses. Finally, it is noted that numerous problems within the scope of this discussion could not be covered, such as other types of damage caused by heat, problems of acclimatization, etc. The reader is referred to the appropriate literature in this regard.

References available on request from the author.

Translated for the National Aeronautics and Space Administration under contract No. NASw-2037 by Techtran Corporation, P.O. Box 729, Glen Burnie, Maryland 21061. Translator: William J. Grimes, M.I.L.